



Do reef corals age?

Bythell, John C.; Brown, Barbara E.; Kirkwood, Thomas B.L.

Published in:
Biological Reviews

DOI:
[10.1111/brv.12391](https://doi.org/10.1111/brv.12391)

Publication date:
2018

Document version
Publisher's PDF, also known as Version of record

Document license:
[CC BY](#)

Citation for published version (APA):
Bythell, J. C., Brown, B. E., & Kirkwood, T. B. L. (2018). Do reef corals age? *Biological Reviews*, 93(2), 1192-1202. <https://doi.org/10.1111/brv.12391>

Do reef corals age?

John C. Bythell^{1,*} , Barbara E. Brown^{1,2} and Thomas B. L. Kirkwood^{3,4}

¹*School of Natural & Environmental Sciences, Ridley Building, Newcastle University, Newcastle upon Tyne NE1 7RU, U.K.*

²*Environmental Research Unit, University of Highlands and Islands, Thurso KW14 7EE, U.K.*

³*Institute for Ageing, Campus for Ageing and Vitality, Newcastle University, Newcastle upon Tyne NE4 5PL, U.K.*

⁴*Center for Healthy Aging, Department of Cellular and Molecular Medicine, University of Copenhagen, Copenhagen 2200, Denmark*

ABSTRACT

Hydra is emerging as a model organism for studies of ageing in early metazoan animals, but reef corals offer an equally ancient evolutionary perspective as well as several advantages, not least being the hard exoskeleton which provides a rich fossil record as well as a record of growth and means of ageing of individual coral polyps. Reef corals are also widely regarded as potentially immortal at the level of the asexual lineage and are assumed not to undergo an intrinsic ageing process. However, putative molecular indicators of ageing have recently been detected in reef corals. While many of the large massive coral species attain considerable ages (>600 years) there are other much shorter-lived species where older members of some populations show catastrophic mortality, compared to juveniles, under environmental stress. Other studies suggestive of ageing include those demonstrating decreased reproduction, increased susceptibility to oxidative stress and disease, reduced regeneration potential and declining growth rate in mature colonies. This review aims to promote interest and research in reef coral ageing, both as a useful model for the early evolution of ageing and as a factor in studies of ecological impacts on reef systems in light of the enhanced effects of environmental stress on ageing in other organisms.

Key words: coral, senescence, ageing, mortality, immortality, determinate growth, indeterminate growth.

CONTENTS

I. Introduction	1192
II. Relevant aspects of coral biology	1193
(1) Evolutionary history	1193
(2) What is the individual?	1193
(3) What are the lifespans of polyps, ramets and genets?	1194
(4) Ontogenic changes in corals	1195
(5) Changes in mortality, reproduction and growth with age	1196
III. How do concepts from biology of ageing apply to corals?	1196
(1) Molecular mechanisms	1196
(2) Ageing in stem cells and germline	1197
(3) Molecular markers of ageing in corals	1197
IV. Environmental change and ageing	1198
V. Conclusions	1199
VI. Acknowledgements	1199
VII. References	1199

I. INTRODUCTION

Generally, a species is regarded as exhibiting ageing, or senescence, if individuals display increasing death rates with age. When this occurs, death is usually preceded by functional

decline affecting most, if not all, aspects of physiology, including reproduction. However there exist species that appear not to age, in the sense that individuals show no tendency either to an increase in death rate or a decline in fertility. The best evidence for absence of ageing is found for

* Address for correspondence (Tel: (+44)(0)191 208 6674); E-mail: john.bythell@ncl.ac.uk

Hydra (Martinez, 1998; Schaible *et al.*, 2015), although similar demographic data have been reported for other species (Finch, 2009; Jones *et al.*, 2014). The classical definition of ageing applies most readily to species where the individual is easily defined, and much of the current uncertainty about ageing in corals derives from questions about the nature of the individual. A further fundamental question concerns the nature of the germline and its relationship to differentiated lineages and structures that might be regarded as soma. The significance of the distinction between germline and soma was first recognised by Weismann, who argued that the germline must be immortal, whereas soma is often subject to ageing and intrinsic mortality (Kirkwood & Cremer, 1982). However, even within the germline, individual cells may deteriorate and die, so the property of immortality belongs to the lineage, not the particular germ cell.

To understand why ageing should have evolved in those species where it is found, it has long been recognised that the central point is that the force of natural selection – that is, its ability to favour or discriminate against alternative alleles – diminishes with increasing age (Hamilton, 1966; Kirkwood & Holliday, 1979). This is because the force of selection at any age is proportional to the fraction of an individual's expected lifetime reproductive output that still remains in its future, which will decline as a consequence of prevailing mortality if nothing else. Not only does this undermine any idea that ageing might have evolved as an actively programmed process of self-destruction, perhaps as a form of population control, but it also has the consequence that natural selection exerts diminishing control over the physiological state of older individuals within the population (Kirkwood & Melov, 2011; Kowald & Kirkwood, 2016).

By combining the significance of the germline/soma distinction together with the recognition that continuing functional integrity of the organism is secured through physiological investments in somatic maintenance and repair, the disposable soma concept (Kirkwood, 1977; Kirkwood & Holliday, 1979; Kirkwood & Austad, 2000) recognises that under pressure of natural selection to make optimal use of metabolic resources, it makes sense only to invest enough in the maintenance of somatic tissues to secure functional integrity during the period that the individual still has a reasonable chance to be alive. For germline, the situation is different, since there is an evolutionary necessity to secure the potential for the lineage to be immortal. Higher investments in maintenance of germ cells are therefore to be expected, and good evidence exists for the down-regulation of maintenance of embryonic stem cells during early differentiation (Saretzki *et al.*, 2004, 2008), for example. The absence of ageing in *Hydra* is therefore most plausibly explained by a ubiquity of germ cells, or similarly highly maintained stem cells throughout the body.

Several important conclusions follow from this logic. Firstly, ageing is caused by the progressive accumulation of damage resulting from the evolved limitation in somatic maintenance. Secondly, the different longevity of species can be explained because exposure to extrinsic mortality

risk varies from one species to another, and consequently selection will favour a higher investment in somatic maintenance in a species better adapted to survive the hazards of its ecological niche than in a species subject to a higher extrinsic level of risk. Thirdly, this evolutionary logic applies similarly to the whole repertoire of mechanisms for cellular maintenance and repair (e.g. DNA damage, oxidative stress, protein denaturation), so it is expected that multiple forms of damage will contribute in parallel, perhaps synergistically, to ageing processes (Kirkwood, 2005b).

Herein, we examine evidence for ageing in corals and consider how progress in understanding ageing in other species may be relevant in advancing research on this question.

II. RELEVANT ASPECTS OF CORAL BIOLOGY

(1) Evolutionary history

Reef-building corals belong to the phylum Cnidaria, class Anthozoa, order Scleractinia and are distinguished from non-reef-building corals by mutualistic and obligate intracellular symbiosis with unicellular dinoflagellate algal symbionts (*Symbiodinium* sp.). While Bridge *et al.* (1995) placed the Anthozoa basally within the phylum Cnidaria, more recent phylogenomic analyses (Schwentner & Bosch, 2015; Zapata *et al.*, 2015) place the common ancestor of the Cnidaria between the Anthozoa and a sister group, the Medusozoa, comprising the Hydrozoa (hydroids, fire corals, siphonophores and their allies) and the Scyphozoa (the familiar jellyfish). There is clearly a deep phylogenetic divergence between Anthozoa and Medusozoa, which likely diverged >500 million years ago (Zapata *et al.*, 2015) so in the context of ageing, hydrozoans such as *Hydra* may not provide an equivalent evolutionary model to anthozoans.

(2) What is the individual?

A key question for ageing of colonial metazoans is what constitutes the individual (Folse & Roughgarden, 2010). The result of sexual reproduction in corals is a ciliated planula larva that settles to the substrate and develops into the primary polyp, or protopolyp. While a few coral species are also able to produce planula larvae via asexual reproduction (Yeoh & Dai, 2010), the majority cannot. Most corals (>80%) are broadcast spawners with external fertilisation and larval development (Harrison, 2011). Except in a few solitary species, the polyps bud to produce a colony. The polyp is a clonal, modular unit that in most species remains physiologically connected to other polyps within the colony via a continuous gastrovascular cavity and associated tissues. While some corals continue to grow in this fashion throughout the life of the colony, many undergo partial mortality that leads to separation of tissues into physiologically isolated sub-colonies or 'ramets'. The total clonal lineage arising from sexual reproduction (the genet)

Table 1. Recorded maximum ages of corals. The majority of these studies have estimated colony age by counting seasonal/annual X-ray density banding in the skeleton (usually with alizarin dye staining or other growth-rate measurements to determine periodicity of density band production). Others estimate age from size using an estimate of historical average annual growth. One (Devlin-Durante *et al.*, 2016) estimates the age of the genet from mutation rate estimates. Thus while genet ages may be substantial, and some colonies can attain ages of several hundred years at least, several studies report relatively short maximum colony ages of a few decades. CAT, computer-assisted tomography.

Species	Age estimate (years)	Region	Age-estimation method	References
<i>Pocillopora verrucosa</i>	3.6–3.9	Micronesia	Isotopic analysis	Richards <i>et al.</i> (2015)
<i>Acropora hemprichii</i>	13–24	Red Sea (northern)	Growth rate \times size	Guzner, Novoplansky & Chadwick (2007)
<i>Coelastrea aspera</i>	15–20	Thailand	Fluorescent density band dating	Scoffin <i>et al.</i> (1997) and Brown <i>et al.</i> (2014)
<i>Coelastrea aspera</i>	20–45	Great Barrier Reef	X-ray density band dating	Babcock (1991)
<i>Goniastrea favulus</i>	20–40	Great Barrier Reef	X-ray density band dating	Babcock (1991)
<i>Porites solida</i>	32	Great Barrier Reef	X-ray density band dating	Potts <i>et al.</i> (1985)
<i>Platygyra sinsensis</i>	50–60	Great Barrier Reef	X-ray density band dating	Babcock (1991)
<i>Porites annae</i>	140	Great Barrier Reef	Growth rate \times size	Connell (1973)
<i>Pavona decussata</i>	150	Japan	Growth rate \times size	Mezaki <i>et al.</i> (2014)
<i>Porites lutea</i>	200	Hong Kong	X-ray density band dating	Goodkin <i>et al.</i> (2011)
<i>Siderastrea siderea</i>	235	Yucatan Peninsula, Mexico	CAT scan and X-ray density band dating	Vasquez-Bedoya <i>et al.</i> (2012)
<i>Diploastrea heliophora</i>	380	Maldives	Growth rate \times size	Schuhmacher, Loch & Loch (2002)
<i>Siderastrea siderea</i>	450	Belize and Bahamas	CAT scan and X-ray density band dating	Saenger <i>et al.</i> (2009)
<i>Porites lobata</i>	677	Great Barrier Reef	X-ray density band dating	Potts <i>et al.</i> (1985)
<i>Acropora palmata</i>	838–2600	Caribbean – selected sites	Somatic mutation rate	Devlin-Durante <i>et al.</i> (2016)
<i>Porites lobata</i>	>1000	Taiwan	Growth rate \times size	Soong <i>et al.</i> (1999)

may comprise many ramets that become widely dispersed, for example during storms, leading to a high degree of clonality within the population (Baums, Miller & Hellberg, 2006; Pinzon *et al.*, 2012; Japaud *et al.*, 2015). Genetic mosaicism has been recorded across the colony due to somatic mutations (van Oppen *et al.*, 2011; Barfield, Aglyamova & Matz, 2016) and chimeric colonies may form by fusion of adjacent, non-clonal colonies, particularly by fusion of juveniles before the immune system is fully developed (Schweinsberg, Tollrian & Lampert, 2016). This implies that cell turnover and migration across the colony, if it occurs (van Oppen *et al.*, 2011), is not extensive or rapid enough to mask the genetic differentiation of the individual polyp lineage.

(3) What are the lifespans of polyps, ramets and genets?

The coral exoskeleton provides a record which can be aged either by counting seasonal–annual density bands or, less accurately, by calculating age from size using an estimated annual growth rate (Buddemeier & Kinzie, 1976). While some individual coral colonies have been estimated to be several hundred years old, up to ~1000 years (Soong, Chen & Chang, 1999), most species lack examples of such ages, suggesting much shorter lifespans (Table 1). Genets may be considerably older than colonies (ramets) and estimates for a relatively fast-growing species, *Acropora palmata*, suggest

genet ages of several hundred to a few thousand years (Devlin-Durante *et al.*, 2016).

‘Coral growth’ commonly refers to the linear extension or mass accretion of the skeleton. However, skeleton growth is not directly related to tissue growth. Coral tissues form a sheet of relatively constant thickness (typically a few millimetres) that expands to cover the surface of the exoskeleton. Maximal tissue growth occurs at sites of increasing surface area and not, necessarily, at sites of high skeletal extension. Indeed Lecointe *et al.* (2016) specifically showed very little cellular proliferation in actively calcifying epithelium of *Stylophora pistillata*.

Darke & Barnes (1993) traced the history of individual coral polyps *via* the growth patterns of their associated corallites (skeletal tubes secreted by the polyp). Polyp ages of the massive coral *Porites* were found to be only 2–3 years on average and a maximum of 5 years, even in colonies that were more than 40 years old. New polyps bud near the apex of mounds on the colony surface and subsequently migrate over time towards depressions on the surface, where they are reabsorbed (Fig. 1). Thus while all the tissues within a genet have the same chronological age since sexual recruitment, the mode of soft tissue growth is likely to influence the number of cell divisions, or ‘replicative age’, experienced by different parts of the colony.

It is often assumed that the tips of branching colony growth forms represent the youngest polyps whereas the basal tissues represent the oldest. However, at least three modes of growth

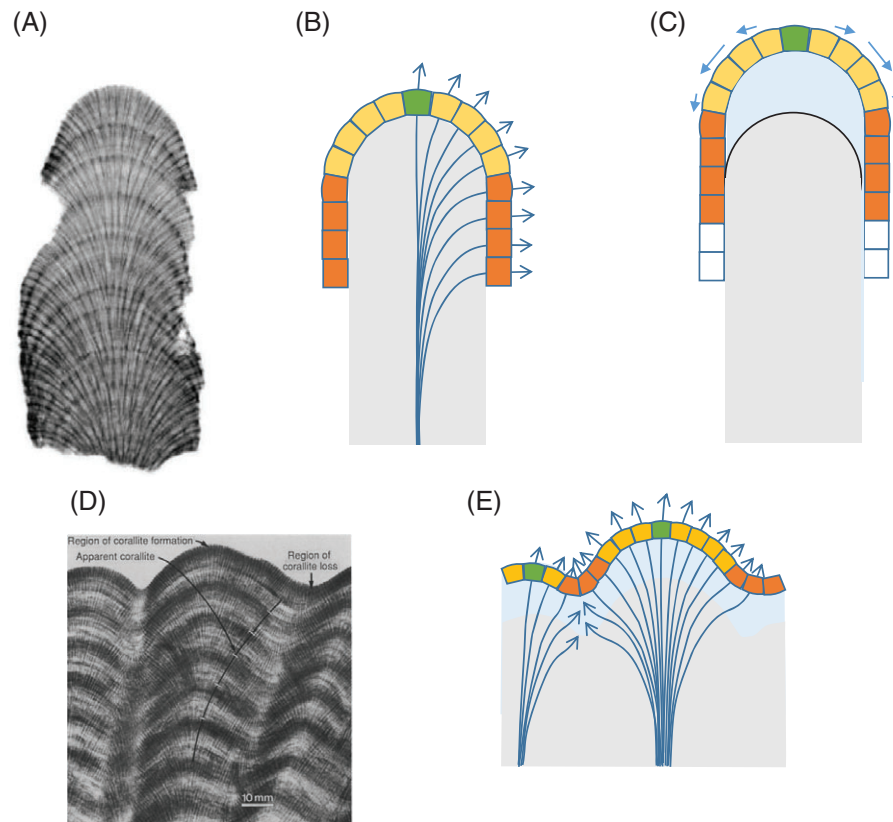


Fig. 1. Models of tissue proliferation based on corallite growth patterns. (A) X-radiograph of columnar coral *Orbicella annularis*, reproduced from Lough & Cooper (2011). (B) In this species, polyps at the apex (green) are the oldest; their corallites can be traced furthest down the column, with younger polyps arising at the periphery of the axis (yellow) and re-orientating their growth outwards (arrows). Tissues in the basal region (orange) do not calcify strongly as the column radius does not expand. (C) Tissues in the peripheral region (yellow), the region of curvature of the column tip, must add new polyps to maintain spacing while the tissue surface area expands as the skeleton is deposited (blue). Polyps at the base must die or be reabsorbed to maintain the relatively constant surface area. (D) X-radiograph of massive *Porites* species reproduced from Darke & Barnes (1993). (E) Corallite growth and tissue proliferation pattern is similar in this species, except that polyps are reabsorbed in depressions and remain in contact with those of adjacent mounds of the colony surface.

have been recognised (Buddemeier & Kinzie, 1976; Rosen, 1986), whereby (i) growth is concentrated in the youngest polyps, (ii) growth occurs equally across all polyps, or (iii) growth is concentrated in the oldest polyps. The common Caribbean coral *Orbicella annularis*, for example, displays the third type of growth, with the oldest polyps occurring at the column apex. Polyps near to the apex undergo rapid proliferation associated with rapid tissue expansion in the area of maximum curvature of the surface, followed by a decline in calcification and cell proliferation lower down the column, and death or reabsorption at the base (Fig. 1A–C). Further studies are clearly needed to assess replicative *versus* chronological age at the polyp and colony scales in relation to the different modes of growth.

(4) Ontogenic changes in corals

Dynamic changes occur in the early life-history stages, ranging from gametes to planulae, and newly settled polyps. These include the establishment of associations with

endosymbiotic algae (where transmission is horizontal and not *via* the parent) (Little, van Oppen & Willis, 2004; Voolstra *et al.*, 2009) and micro-organisms (Sharp *et al.*, 2010; Apprill *et al.*, 2012; Sharp, Distel & Paul, 2012), and the development of immunity and allorecognition systems (Frank *et al.*, 1997; Nozawa & Loya, 2005; Puill-Stephan *et al.*, 2012). Physiological changes continue to be made as corals develop from juveniles to adults and through adulthood with the following examples relating to both ramets and genets, depending on the study. These changes include alterations in gene expression (Reyes-Bermudez *et al.*, 2009, 2016) and reproductive status, with corals delaying becoming reproductive for up to 8–10 years in some cases (Harrison & Wallace, 1990; Wallace, 1999) although many shorter-lived corals become reproductive earlier (1–2 years) (Harrison & Wallace, 1990; Hall & Hughes, 1996). Stable associations with endosymbiotic algae form in adulthood that differ from those of juveniles (Byler *et al.*, 2013; Reich, Robertson & Goodbody-Gringley, 2017), a process taking up to 3.5 years in some species (Abrego, Van Oppen & Willis, 2009). Similarly, associations

with micro-organisms appear to be age dependent with differences in microbial consortia noted between juveniles and adults (Littman *et al.*, 2009; Williams *et al.*, 2015) and between adults of 3–12 years in age (Williams *et al.*, 2015). A greater investment of energy into tissue growth has been reported in small corals compared with skeletal-dominated growth in larger corals (Anthony, Connolly & Willis, 2002). Skeletal growth patterns also alter, with rate of branch formation in *Pocillopora damicornis* decreasing with age (Permata & Hidaka, 2005). Energy budgets in older corals must continually readjust to increased demands of reproduction, growth, tissue and skeletal repair (Philipp & Fabricius, 2003; Anthony *et al.*, 2009; Pisapia, Anderson & Pratchett, 2014).

(5) Changes in mortality, reproduction and growth with age

Corals have long been considered non-ageing with respect to age-specific mortality and reproduction (Flatt, 2012). Initial larval mortality is high (Graham, Baird & Connolly, 2008) and younger colonies suffer greater total mortality than older colonies under natural conditions (Connell, 1973; Babcock, 1991; Sakai, 1998). Conversely, incidence of partial mortality is higher in older colonies (Hughes & Jackson, 1985; Babcock, 1991; Bythell, Gladfelter & Bythell, 1993). Partial mortality can be significant, accounting for a greater proportion of tissue loss within the population than whole-colony mortality (Bythell *et al.*, 1993; Baird & Marshall, 2002). However, colony size may not reflect age, as partial mortality and fragmentation may cause reductions in colony size (Hughes & Jackson, 1980) and greater levels of partial mortality in larger colonies may be explained as a probabilistic process; larger colonies being more likely to encounter damage, but less likely to be killed by it (Hughes & Jackson, 1985).

The fecundity of a colony normally increases with age as a result of increased number of polyps and/or increased polyp fecundity (Szmant, 1986; Harrison & Wallace, 1990). Few studies have investigated age-related polyp fecundity (where age was established through growth-rate measurements deduced by alizarin staining and density banding patterns) apart from those of Babcock (1991) and Sweet *et al.* (2017), where special efforts were made to consider genets rather than ramets. Babcock (1991) showed that polyp fecundity in three coral species on the Great Barrier Reef (GBR) increased sigmoidally during an extended period of adolescence and reached a stable asymptote after 10–15 years. A similar pattern was noted for one of these species in western Thailand although the asymptote was reached within 4–5 years (Sweet *et al.*, 2017). It should be noted however that Babcock (1991) deliberately biased sampling towards small- to medium-sized corals, his aim being to establish the earliest age/size at which the corals became reproductive. In Thailand incidence of aged corals was also low because of catastrophic mortality in an earlier bleaching event (Brown *et al.*, 2014). Uncertainty about the reproductive status of the oldest corals merits further study, particularly since Babcock (1991) has been cited as providing the ‘best evidence of negative senescence

in any animal species’ – where ‘negative senescence’ is characterised by an *increase* in fertility as mortality declines with age (Vaupel *et al.*, 2004).

While older corals may remain sexually active (Mezaki, Keshavmurthy & Chen, 2014) with relatively high polyp fecundity (Babcock, 1991; Wallace, 1999; Sweet *et al.*, 2017) current data do not rule out reproductive decline as colonies age. Reproductive senescence has been suggested to explain declining polyp fecundity either in the colony as a whole (Rinkevich & Loya, 1986) or in aged parts (Soong & Lang, 1992; Irikawa *et al.*, 2011; Nozawa & Lin, 2014). Conversely, Hall & Hughes (1996) showed colony fecundity increasing with size in all species, although there was no relationship between polyp fecundity and colony size. This work has been cited as evidence for a lack of senescence (Wallace, 1999), but few corals lived longer than 10 years at their study site. Also, although they avoided the marginal parts of the coral, it is not clear whether they sampled the oldest polyps.

The question of whether corals exhibit indeterminate growth (invariant growth rate with age) or determinate growth (declining growth rate with age) has been debated since the earliest review of coral growth (Buddemeier & Kinzie, 1976). Their review concluded that some species such as *Manicina areolata*, *Fungia scutaria* and *Pocillopora meandrina* showed determinate growth but many of the long-lived, massive species such as those of the genus *Porites* showed indeterminate growth with no systematic decrease in skeletal extension over decades (Buddemeier & Kinzie, 1976).

Marked decline in calcification rate was shown to occur in *Stylophora pistillata* 3–6 months before partial and then full mortality (Rinkevich & Loya, 1986) and perturbed growth of tissues and skeleton have been noted in the oldest parts of plating *Acropora cytherea* colonies (Irikawa *et al.*, 2011). Meesters & Bak (1995) described decreased lesion regeneration capability along the length of branches of *Acropora palmata* which they ascribed to polyp age, while Elahi & Edmunds (2007) similarly observed significantly lower calcification rates in explants taken from older parts of *Madracis mirabilis* colonies that were returned to the field after manipulation and their growth monitored over 3 months.

Declines in reproduction and/or growth with age (or size) is therefore evident in some species where it is manifest at the level of the polyp, colony and/or part of the colony. Examples have mostly been reported in ‘short-lived’ species such as *Stylophora pistillata* and *Pocillopora damicornis* but detailed polyp–colony-level studies are required across a broader range of lifespans and across the full spectrum of ages.

III. HOW DO CONCEPTS FROM BIOLOGY OF AGEING APPLY TO CORALS?

(1) Molecular mechanisms

It is generally accepted that ageing is driven by progressive accumulation of molecular and cellular defects, leading eventually to functional impairments, chronic diseases and

death. Various ‘hallmarks’ of ageing include, at the molecular level genome instability, erosion of telomeres, mitochondrial dysfunction and protein denaturation, and at the cellular level replicative senescence, apoptosis, inflammatory responses, stem-cell deterioration and impaired intercellular communication (Lopez-Otin *et al.*, 2013).

The multiplicity of ageing mechanisms presents significant challenges for determining the ultimate causes of intrinsic ageing. While there is evidence that somatic nuclear mutations, shortened telomeres, oxidative damage and defective mitochondria all accumulate across the life course, the accumulations of individual forms of damage are generally too small for ageing to be explicable in terms of any single mechanism. There is therefore growing recognition that ageing needs to be understood in terms of its ‘systems biology’, in which contributions of different mechanisms act synergistically (Kirkwood, 2011). Furthermore, some ageing hallmarks, e.g. stem-cell deterioration, may be secondary consequences of others, e.g. genome instability. Processes such as apoptosis, replicative senescence and inflammation, all of which play a part in ageing, actually had their evolutionary origins as adaptations conferring benefits during earlier life stages. For example, apoptosis serves variously to aid morphogenesis and to delete auto-reactive lymphocytes as well as to remove cells which suffer potentially carcinogenic damage. Such a response, which is adaptive in a younger organism when damaged cells are relatively rare, may become maladaptive in an older organism when damage becomes pervasive. This capacity for damage-response mechanisms to acquire pro-ageing properties most plausibly explains why ageing shows some features of active regulation, even though ageing is not believed to be programmed in its own right.

(2) Ageing in stem cells and germline

The kinds of damage contributing to somatic ageing mostly also arise in germ cells. The fact that the germline is immortal does not mean that individual germ cells do not suffer damage and die, and of course many of them do. But the lineage continues and this means it contains a sequence of cells that does not progressively accumulate damage. How the germline sustains itself is thus a question of fundamental importance.

Firstly, it may be that germ cells invest more heavily in maintenance and repair (Kirkwood, 1977). Secondly, within germline there tends to be opportunity for selection to act as a means of ‘quality assurance’. The over-production of gametes, from which only the fittest contribute to reproductive success, and a cellular organisation of the germline which is adapted to maximise the screening out of defects are important factors in this regard.

Tissue stem cells occupy a position intermediate to germline and terminally differentiated somatic cells. Stem cells support long-term renewal processes in many tissues and are capable of prolonged survival, but they are not immune to age-related deterioration. In mammals decreased regenerative capacity, as a result of decline in replicative

function of certain stem cell types, contributes to ageing (Sharpless & Depinho, 2007). Most observations on stem cells in Cnidaria have been made in *Hydra* where their functions include the ability to regenerate somatic tissues and germ cell lines. While regeneration of damaged tissues has been widely documented in corals, nothing is known about the function of stem cells in this process, or indeed, whether they even exist in anthozoans (Rinkevich, Matranga & Rinkevich, 2007).

(3) Molecular markers of ageing in corals

The literature on molecular markers of ageing in corals is both recent and restricted. Given the scope for asexually generated coral colonies to achieve considerable age, it is unsurprising that significant somatic mutation can accumulate across the colony. This was regarded by van Oppen *et al.* (2011) as a potentially important contributor to adaptation and evolution of reef corals, since germ-cell differentiation was believed to occur continuously from somatic stem cells (Buss, 1983). However, Barfield *et al.* (2016) recently detected somatic mutations across large colonies of the reef coral *Orbicella faveolata*, which were absent in the germline, implying a segregation of germline stem cells from somatic stem cells, as seen in the majority of the Metazoa.

Telomeres have received much attention as molecular markers of ageing since gradual loss of telomere repeats occurs in human differentiated somatic cells, where telomerase is generally inactivated, contributing to replicative senescence (Aubert, Hills & Lansdorp, 2012). Sköld & Obst (2011) showed that parental strains of a colonial ascidian showed both significantly lower telomerase activity and shorter telomeres compared to the offspring, leading the authors to conclude that the ascidian had not escaped ageing and that only sexual reproduction allowed total rejuvenation.

Several preliminary studies describe telomere characteristics in corals (Sinclair, Richmond & Ostrander, 2007; Zielke & Bodnar, 2010; Nakamichi *et al.*, 2012) (Table 2). For the short-lived branching coral *Acropora digitifera* telomere length was significantly longer in sperm than in the planulae which was in turn longer than in adult polyps (Tsuta *et al.*, 2014). However, in another species, *Galaxea fascicularis*, no significant differences were found (Tsuta & Hidaka, 2013). Tsuta *et al.* (2014) proposed that this may be explained by *G. fascicularis* having a longer lifespan, perhaps associated with a low rate of telomere change due to the high levels of telomerase activity noted in adults (Nakamichi *et al.*, 2012).

Telomere shortening has also been reported in the solitary coral *Ctenactis echinata*, which is not known to divide asexually (Ojimi, Loya & Hidaka, 2012). However, while differences were observed in telomere length between sperm and somatic tissues, no significant effects of age were seen in adults. Further research is needed to establish appropriate methodologies for telomere length estimation in a wide range of coral species of different ages by accounting for potential analytical and interpretative pitfalls (Aubert *et al.*, 2012; Nussey *et al.*, 2014) as well as considering the differences

Table 2. Telomere restriction fragment (TRF) lengths (kb) for selected coral material and symbiotic algae. Sample size (N) and standard deviation (SD) indicated where cited.

Species/clade	N	TRF length (kb) (\pm SD)	References
<i>Agaricia fragilis</i>	9	19.2 \pm 1.1	Zielke & Bodnar (2010)
<i>Madracis aurentenra</i>	5	19.2 \pm 0.8	
<i>Acropora millepora</i> (gametes)	3	21.0 \pm 0.4	Sinclair <i>et al.</i> (2007)
<i>Acropora surculosa</i>		4.0	
<i>Dipsastraea pallida</i>		\sim 9	
<i>Leptoria phrygia</i>		\sim 9	
<i>Acropora digitifera</i> (sperm)	11	16.1 \pm 2.7	Tsuta <i>et al.</i> (2014)
<i>Acropora digitifera</i> (planulae)	8	11.7 \pm 3.4	
<i>Acropora digitifera</i> (polyps)	10	8.9 \pm 1.8	Tsuta & Hidaka (2013)
<i>Galaxea fascicularis</i> (sperm)	2	17.9	
<i>Galaxea fascicularis</i> (planulae)	3	7.5 \pm 2.7	
<i>Galaxea fascicularis</i> (polyps)	6	15.6 \pm 3.9	
<i>Symbiodinium</i> Clade B cultured	4	6.6 \pm 0.2	Zielke & Bodnar (2010)
<i>Symbiodinium</i> Clade C cultured	4	2.4 \pm 0.2	
<i>Symbiodinium</i> Clade A cultured	4	<1.0	
<i>Symbiodinium</i> (freshly isolated)		>20.0	

in replicative age across the landscape of the coral colony discussed in Section 2.3.

Somatic mutation and DNA damage have also been cited as important contributors to ageing (Kirkwood, 2005a; Chen, Hales & Ozanne, 2007; Garinis *et al.*, 2008; White *et al.*, 2015). Interestingly, somatic mutations have been estimated to occur with a high frequency in corals, with as many as \sim 100 million mutations arising in a typical 30 cm diameter *Acropora millepora* colony (van Oppen *et al.*, 2011), possibly a conservative estimate as no account was made of cell renewal. Phenotypically observable mutation rate is similarly high, with 300–500 mutations estimated to accumulate over the lifetime of the colony in *Orbicella faveolata* (Barfield *et al.*, 2016). High frequencies of somatic mutations have also been proposed to explain significant intra-colonial variability observed in other coral species (Maier *et al.*, 2012; Schweinsberg *et al.*, 2015, 2016). In addition, significant levels of somatic mutation have been reported in microsatellite loci between ramets within the genet of *A. palmata* from sites across the Caribbean (Devlin-Durante *et al.*, 2016). Thus a number of recent studies highlight a high frequency of somatic mutations, and while some may be advantageous, deleterious mutations will likely also accumulate throughout the coral's lifetime.

Reactive oxygen species (ROS) have been heavily implicated in ageing (Kourtis & Tavernarakis, 2011), although a direct connection between oxidative damage and ageing is debatable (Speakman *et al.*, 2015). Corals harbouring photosynthetic symbiotic algae are routinely challenged by ROS and have evolved strong defences, including enzymatic antioxidants, a suite of non-enzymatic ROS scavengers and ultraviolet-absorbing compounds (Lesser, 2011). However, these defences may be overcome during periods of elevated temperature stress, leading to coral bleaching and mortality. Bleaching is a response to the combined stresses of temperature and irradiance, where the coral pales as a result of loss of symbiotic zooxanthellae and/or their pigments

(Brown, 1997), and its increasing occurrence all around the world is placing corals under pressure (Rinkevich, Avishai & Rabinowitz, 2005; Lesser, 2011).

IV. ENVIRONMENTAL CHANGE AND AGEING

Several studies have documented greater vulnerability of adult compared to juvenile corals during mass-bleaching events (Hoeksema, 1991; Mumby, 1999; Normile, 2000; Edwards *et al.*, 2001; Loya *et al.*, 2001; Bena & van Woesik, 2004; Brandt, 2009; Phongsuwan & Chansang, 2012; Depczynski *et al.*, 2013; Brown *et al.*, 2014). In some cases this has been attributed to the cryptic nature of juveniles which may reduce light stress (Hoeksema, 1991; Mumby, 1999). However, there are other examples where juveniles do not have such protection but are still more thermally tolerant (Loya *et al.*, 2001; Brown *et al.*, 2014). Another possible factor is the rate of passive diffusion being greater in smaller, less morphologically complex, younger colonies (Loya *et al.*, 2001; Nakamura & van Woesik, 2001). However, this also may not apply in all cases, with flow patterns being highly complex in natural reef environments (Hench & Rosman, 2013). For example, Brown *et al.* (2014) proposed that senescence may have played a role in the demise of older corals of the 'short-lived' intertidal coral *Coelastrea aspera* living close to its thermal limits on the reef flat at Phuket, Thailand in 2010, when stressful sea temperatures over 32°C prevailed for 7 weeks. Earlier demographic studies showed that the population was mainly composed of older, larger colonies which recruited in the early 1990s. Following severe bleaching, all colonies >8 cm diameter showed extensive partial mortality with 25% of those >20 cm diameter being completely killed. By contrast, colonies <8 cm diameter, all of which exhibited 100% bleaching, recovered completely. In this case, the potential ameliorating shading effects due to

occupation of cryptic habitats by recently settled corals was discounted as an explanation, since juvenile *C. aspera* colonies were not found in shaded habitats but on open reef surfaces exposed to full irradiances. In terms of water flow, the smooth contours of hemispherical massive corals such as *C. aspera* would not increase frictional drag and mass transfer potential in the same way as that described for branching corals (Nakamura & van Woesik, 2001), with massive corals having a higher mass transfer rate than branching species (van Woesik *et al.*, 2012). In the field, spatial flow patterns at the colony level, on a shallow reef flat composed of hemispherical colonies, are extremely complex and where colonies are densely spaced, as in the present example, inertial forces can significantly reduce amplitude variations between colonies with higher water flows around larger colonies compared with smaller ones (Hench & Rosman, 2013; J. L. Hench, personal communication). It is also interesting to note that juvenile *C. aspera* showed a significantly greater enhancement of antioxidant enzymes and heat-shock proteins compared with adults in short-term elevated temperature experiments (B. E. Brown, unpublished data). Whether this response is due to greater energetic constraints of older, reproductive corals or to age-related deterioration is unknown. Should senescence play a role in the demise of this species, it is possible that increased temperature anomalies could exacerbate physiological deterioration – a view also raised by Irikawa *et al.* (2011) who noted that growth anomalies on the oldest parts of *A. cytherea* increased in response to increasing environmental temperature stresses.

A further intriguing possibility is that bleaching may be a host innate immune response to a compromised symbiont (Weis, 2008). If environmental changes impose increased stress on the system, this may activate a response that in normal circumstances serves to protect against an acute challenge. If so, an important parallel could be drawn to mammalian ageing, where gradual accumulation of damage results in chronic activation of innate immune responses that in other circumstances would serve to protect against acute threats (e.g. infection, wounding) but which in the context of ageing may simply exacerbate the overall deterioration in homeostasis.

V. CONCLUSIONS

(1) The issue of whether, and to what extent, ageing occurs in reef corals is timely to reconsider in the light of both new developments in the biology of ageing, and growing appreciation of threats to long-term survival of corals through environmental stresses. Corals occupy an intriguing position at the boundary between species where there is a clear germline/soma distinction and ageing obviously does occur, and others such as *Hydra* where such a distinction is less well defined and ageing appears absent. The fact that ageing itself is now understood to result from accumulation of molecular and cellular damage, much of which can be attributed to a complex of intrinsic and extrinsic stressors, makes

the potential connections in the case of corals particularly interesting.

(2) The established dogma that corals are non-ageing seems increasingly questionable in view of findings such as the distinction of the germline and soma, the potential for high somatic mutation rates, detection of ageing markers as well as reproductive decline, decreased growth and increased susceptibility to stress in older corals.

(3) Should ageing occur in some coral species there is potential for interactions between age-related decline and environmental impacts such as those due to climate change, with differential effects on survivorship and fitness. Loss of older and larger colonies with high fecundity would have immediate negative impacts on reef community structure (Potts *et al.*, 1985) and structural complexity (Madin & Connolly, 2006; Madin, Hughes & Connolly, 2012).

(4) Ageing could also influence the interpretation of methodologies routinely employed to assess effects of climate change on corals such as worldwide growth rate changes (De'ath, Fabricius & Lough, 2013; Ridd, da Silva & Stieglitz, 2013); reconstruction of coral environmental history using isotopic and geochemical signals (McConnaughey, 1989; Darke & Barnes, 1993; DeLong *et al.*, 2016), use of colonies at different life stages in studies where inherent variability in responses have been noted (Sweet & Brown, 2016) and even the selection of tissues from colonies where polyps of different ages are contained within 'nubbins' (coral colony fragments) used in experimental assays.

(5) Reef corals provide a comparative system to explore the early evolution of ageing in animals, with both similarities and distinct differences to *Hydra* and its allies. Belonging to a sister taxon, the Anthozoa, that diverged very early (>500 million years ago) from the Medusozoa, there are both solitary and colonial growth forms and a great range of lifespans from decades to up to thousands of years, suggesting distinct ageing responses. The ability to track development and ages of individual polyps, as well as annual growth records of the colony *via* the calcium carbonate exoskeleton (Buddemeier & Kinzie, 1976; Darke & Barnes, 1993) is a significant advantage in reef corals. We highlight here several areas that need urgent attention to allow the development of reefs corals as a model system for ageing research.

VI. ACKNOWLEDGEMENTS

We would like to thank Dr Brian Rosen for helpful discussions on growth patterns in reef corals. B.E.B. acknowledges the support of the Leverhulme Trust (Grant EM-2013-058) and J.C.B. acknowledges support of the Natural Environment Research Council (NE/H020616).

VII. REFERENCES

ABREGO, D., VAN OPPEN, M. J. H. & WILLIS, B. L. (2009). Highly infectious symbiont dominates initial uptake in coral juveniles. *Molecular Ecology* **18**, 3518–3531.

- ANTHONY, K. R. N., CONNOLLY, S. R. & WILLIS, B. L. (2002). Comparative analysis of energy allocation to tissue and skeletal growth in corals. *Limnology and Oceanography* **47**, 1417–1429.
- ANTHONY, K. R. N., HOOGENBOOM, M. O., MAYNARD, J. A., GROTTOLI, A. G. & MIDDLEBROOK, R. (2009). Energetics approach to predicting mortality risk from environmental stress: a case study of coral bleaching. *Functional Ecology* **23**, 539–550.
- APPRILL, A., MARLOW, H. Q., MARTINDALE, M. Q. & RAPPE, M. S. (2012). Specificity of associations between bacteria and the coral *Pocillopora meandrina* during early development. *Applied and Environmental Microbiology* **78**, 7467–7475.
- AUBERT, G., HILLS, M. & LANSDORP, P. M. (2012). Telomere length measurement—Caveats and a critical assessment of the available technologies and tools. *Mutation Research—Fundamental and Molecular Mechanisms of Mutagenesis* **730**, 59–67.
- BABCOCK, R. C. (1991). Comparative demography of 3 species of scleractinian corals using age-dependent and size-dependent classifications. *Ecological Monographs* **61**, 225–244.
- BAIRD, A. H. & MARSHALL, P. A. (2002). Mortality, growth and reproduction in scleractinian corals following bleaching on the Great Barrier Reef. *Marine Ecology Progress Series* **237**, 133–141.
- BARFIELD, S., AGLYAMOVA, G. V. & MATZ, M. V. (2016). Evolutionary origins of germline segregation in Metazoa: evidence for a germ stem cell lineage in the coral *Orbicella faveolata* (Cnidaria, Anthozoa). *Proceedings of the Royal Society B: Biological Sciences* **283**, 20152128.
- BAUMS, I. B., MILLER, M. W. & HELLBERG, M. E. (2006). Geographic variation in clonal structure in a reef-building Caribbean coral, *Acropora palmata*. *Ecological Monographs* **76**, 503–519.
- BENA, C. & VAN WOESIK, R. (2004). The impact of two bleaching events on the survival of small coral colonies (Okinawa, Japan). *Bulletin of Marine Science* **75**, 115–125.
- BRANDT, M. E. (2009). The effect of species and colony size on the bleaching response of reef-building corals in the Florida Keys during the 2005 mass bleaching event. *Coral Reefs* **28**, 911–924.
- BRIDGE, D., CUNNINGHAM, C. W., DESALLE, R. & BUSS, L. W. (1995). Class-level relationships in the phylum Cnidaria – molecular and morphological evidence. *Molecular Biology and Evolution* **12**, 679–689.
- BROWN, B. E. (1997). Coral bleaching: causes and consequences. *Coral Reefs* **16**, S129–S138.
- BROWN, B. E., DUNNE, R. P., PHONGSUWAN, N., PATCHIM, L. & HAWKRIDGE, J. M. (2014). The reef coral *Goniastrea aspera*: a 'winner' becomes a 'loser' during a severe bleaching event in Thailand. *Coral Reefs* **33**, 395–401.
- BUDDEMEIER, R. W. & KINZIE, R. A. (1976). Coral growth. *Oceanography and Marine Biology Annual Review* **14**, 183–225.
- BUSS, L. W. (1983). Evolution, development, and the units of selection. *Proceedings of the National Academy of Sciences of the United States of America—Biological Sciences* **80**, 1387–1391.
- BYLER, K. A., CARMÍ-VEAL, M., FINE, M. & GOULET, T. L. (2013). Multiple symbiont acquisition strategies as an adaptive mechanism in the coral *Stylophora pistillata*. *PLoS One* **8**, e59596.
- BYTHELL, J. C., GLADFELTER, E. H. & BYTHELL, M. (1993). Chronic and catastrophic natural mortality of three common Caribbean reef corals. *Coral Reefs* **12**, 143–152.
- CHEN, J. H., HALES, C. N. & OZANNE, S. E. (2007). DNA damage, cellular senescence and organismal ageing: causal or correlative? *Nucleic Acids Research* **35**, 7417–7428.
- CONNELL, J. H. (1973). Population ecology of reef-building corals. In *Biology and Geology of Coral Reefs*, Volume 2. *Biology* (eds O. A. JONES and R. ENDEAN), pp. 205–245. Academic Press, New York.
- DARKE, W. M. & BARNES, D. J. (1993). Growth trajectories of corallites and ages of polyps in massive colonies of reef-building corals of the genus *Porites*. *Marine Biology* **117**, 321–326.
- DE'ATH, G., FABRICIUS, K. & LOUGH, J. (2013). Yes – Coral calcification rates have decreased in the last twenty-five years! *Marine Geology* **346**, 400–402.
- DELONG, K. L., MAUPIN, C. R., FLANNERY, J. A., QUINN, T. M. & SHEN, C. C. (2016). Refining temperature reconstructions with the Atlantic coral *Siderastrea sidera*. *Palaeogeography Palaeoclimatology Palaeoecology* **462**, 1–15.
- DEPCZYNSKI, M., GILMOUR, J. P., RIDGWAY, T., BARNES, H., HEYWARD, A. J., HOLMES, T. H., MOORE, J. A. Y., RADFORD, B. T., THOMSON, D. P., TINKLER, P. & WILSON, S. K. (2013). Bleaching, coral mortality and subsequent survivorship on a West Australian fringing reef. *Coral Reefs* **32**, 233–238.
- DEVLIN-DURANTE, M. K., MILLER, M. W., PRECHT, W. F., BAUMS, I. B. & Caribbean Acropora Research Group (2016). How old are you? Genet age estimates in a clonal animal. *Molecular Ecology* **25**, 5628–5646.
- EDWARDS, A. J., CLARK, S., ZAHIR, H., RAJASURIYA, A., NASEER, A. & RUBENS, J. (2001). Coral bleaching and mortality on artificial and natural reefs in Maldives in 1998, sea surface temperature anomalies and initial recovery. *Marine Pollution Bulletin* **42**, 7–15.
- ELAHI, R. & EDMUNDS, P. J. (2007). Tissue age affects calcification in the Scleractinian coral *Madracis mirabilis*. *Biological Bulletin* **212**, 20–28.
- FINCH, C. E. (2009). Update on slow aging and negligible senescence – a mini-review. *Gerontology* **55**, 307–313.
- FLATT, T. (2012). A new definition of aging? *Frontiers in Genetics* **3**, 148.
- FOLSE, H. J. & ROUGHGARDEN, J. (2010). What is an individual organism? A multilevel selection perspective. *Quarterly Review of Biology* **85**, 447–472.
- FRANK, U., OREN, U., LOYA, Y. & RINKEVICH, B. (1997). Alloimmune maturation in the coral *Stylophora pistillata* is achieved through three distinctive stages, 4 months post-metamorphosis. *Proceedings of the Royal Society B: Biological Sciences* **264**, 99–104.
- GARINIS, G. A., VAN DER HORST, G. T. J., VIJG, J. & HOEIJMAKERS, J. H. J. (2008). DNA damage and ageing: new-age ideas for an age-old problem. *Nature Cell Biology* **10**, 1241–1247.
- GOODKIN, N. F., SWITZER, A. D., MCCORRY, D., DEVANTIER, L., TRUE, J. D., HUGHEN, K. A., ANGELINE, N. & YANG, T. T. (2011). Coral communities of Hong Kong: long-lived corals in a marginal reef environment. *Marine Ecology Progress Series* **426**, 185–196.
- GRAHAM, E. M., BAIRD, A. H. & CONNOLLY, S. R. (2008). Survival dynamics of scleractinian coral larvae and implications for dispersal. *Coral Reefs* **27**, 529–539.
- GUZNER, B., NOVOPOLANSKY, A. & CHADWICK, N. E. (2007). Population dynamics of the reef-building coral *Acropora hemprichii* as an indicator of reef condition. *Marine Ecology Progress Series* **333**, 143–150.
- HALL, V. R. & HUGHES, T. P. (1996). Reproductive strategies of modular organisms: comparative studies of reef-building corals. *Ecology* **77**, 950–963.
- HAMILTON, W. D. (1966). The moulding of senescence by natural selection. *Journal of Theoretical Biology* **12**, 12–45.
- HARRISON, P. L. (2011). Sexual reproduction of scleractinian corals. In *Coral Reefs: An Ecosystem in Transition* (eds Z. DUBINSKY and N. STAMBLER), pp. 59–85. Springer, Berlin.
- HARRISON, P. L. & WALLACE, C. C. (1990). Reproduction, dispersal and recruitment of scleractinian corals. In *Ecosystems of the World: Coral Reefs* (ed. Z. DUBINSKY), pp. 133–207. Elsevier, Amsterdam.
- HENCH, J. L. & ROSMAN, J. H. (2013). Observations of spatial flow patterns at the coral colony scale on a shallow reef flat. *Journal of Geophysical Research—Oceans* **118**, 1142–1156.
- HOEKSEMA, B. W. (1991). Control of bleaching in mushroom coral populations (Scleractinia, Fungiidae) in the Java Sea – stress tolerance and interference by life-history strategy. *Marine Ecology Progress Series* **74**, 225–237.
- HUGHES, T. P. & JACKSON, J. B. C. (1980). Do corals lie about their age? Some demographic consequences of partial mortality, fission, and fusion. *Science* **209**, 713–715.
- HUGHES, T. P. & JACKSON, J. B. C. (1985). Population dynamics and life histories of foliaceous corals. *Ecological Monographs* **55**, 141–166.
- IRIKAWA, A., CASARETO, B. E., SUZUKI, Y., AGOSTINI, S., HIDAKA, M. & VAN WOESIK, R. (2011). Growth anomalies on *Acropora cytherea* corals. *Marine Pollution Bulletin* **62**, 1702–1707.
- JAPAUD, A., BOUCHON, C., MANCEAU, J. L. & FAUVELOT, C. (2015). High clonality in *Acropora palmata* and *Acropora cervicornis* populations of Guadeloupe, French Lesser Antilles. *Marine and Freshwater Research* **66**, 847–851.
- JONES, O. R., SCHEUERLEIN, A., SALGUERO-GOMEZ, R., CAMARDA, C. G., SCHAIKLE, R., CASPER, B. B., DAHLGREN, J. P., EHRLER, J., GARCIA, M. B., MENGES, E. S., QUINTANA-ASCENCIO, P. F., CASWELL, H., BAUDISCH, A. & VAUPEL, J. W. (2014). Diversity of ageing across the tree of life. *Nature* **505**, 169–173.
- KIRKWOOD, T. B. L. (1977). Evolution of aging. *Nature* **270**, 301–304.
- KIRKWOOD, T. B. L. (2005a). Time of our lives – What controls the length of life? *EMBO Reports* **6**, S4–S8.
- KIRKWOOD, T. B. L. (2005b). Understanding the odd science of aging. *Cell* **120**, 437–447.
- KIRKWOOD, T. B. L. (2011). Systems biology of ageing and longevity. *Philosophical Transactions of the Royal Society B: Biological Sciences* **366**, 64–70.
- KIRKWOOD, T. B. L. & AUSTAD, S. N. (2000). Why do we age? *Nature* **408**, 233–238.
- KIRKWOOD, T. B. L. & CREMER, T. (1982). Cytogerontology since 1881 – a reappraisal of August Weismann and a review of modern progress. *Human Genetics* **60**, 101–121.
- KIRKWOOD, T. B. L. & HOLLIDAY, R. (1979). Evolution of aging and longevity. *Proceedings of the Royal Society of London Series B—Biological Sciences* **205**, 531–546.
- KIRKWOOD, T. B. L. & MELOV, S. (2011). On the programmed/non-programmed nature of ageing within the life history. *Current Biology* **21**, R701–R707.
- KOURTIS, N. & TAVERNARAKIS, N. (2011). Cellular stress response pathways and ageing: intricate molecular relationships. *EMBO Journal* **30**, 2520–2531.
- KOWALD, A. & KIRKWOOD, T. B. L. (2016). Can aging be programmed? A critical literature review. *Ageing Cell* **15**, 986–998.
- LECOINTE, A., DOMART-COULON, I., PARIS, A. & MEIBOM, A. (2016). Cell proliferation and migration during early development of a symbiotic scleractinian coral. *Proceedings of the Royal Society B: Biological Sciences* **283**, 20160206.
- LESSER, M. P. (2011). Coral bleaching: causes and mechanisms. In *Coral Reefs: An Ecosystem in Transition* (eds Z. DUBINSKY and N. STAMBLER), pp. 405–419. Springer, Dordrecht Netherlands.
- LITTLE, A. F., VAN OPPEN, M. J. H. & WILLIS, B. L. (2004). Flexibility in algal endosymbioses shapes growth in reef corals. *Science* **304**, 1492–1494.
- LITTMAN, R. A., WILLIS, B. L., PFEFFER, C. & BOURNE, D. G. (2009). Diversities of coral-associated bacteria differ with location, but not species, for three acroporid corals on the Great Barrier Reef. *FEMS Microbiology Ecology* **68**, 152–163.

- LOPEZ-OTIN, C., BLASCO, M. A., PARTRIDGE, L., SERRANO, M. & KROEMER, G. (2013). The hallmarks of aging. *Cell* **153**, 1194–1217.
- LOUGH, J. M. & COOPER, T. F. (2011). New insights from coral growth band studies in an era of rapid environmental change. *Earth-Science Reviews* **108**, 170–184.
- LOYA, Y., SAKAI, K., YAMAZATO, K., NAKANO, Y., SAMBALI, H. & VAN WOESIK, R. (2001). Coral bleaching: the winners and the losers. *Ecology Letters* **4**, 122–131.
- MADIN, J. S. & CONNOLLY, S. R. (2006). Ecological consequences of major hydrodynamic disturbances on coral reefs. *Nature* **444**, 477–480.
- MADIN, J. S., HUGHES, T. P. & CONNOLLY, S. R. (2012). Calcification, storm damage and population resilience of tabular corals under climate change. *PLoS One* **7**, e46637.
- MAIER, E., BUCKENMAIER, A., TOLLRIAN, R. & NURNBERGER, B. (2012). Intracolony genetic variation in the scleractinian coral *Seriatopora hystrix*. *Coral Reefs* **31**, 505–517.
- MARTINEZ, D. E. (1998). Mortality patterns suggest lack of senescence in *Hydra*. *Experimental Gerontology* **33**, 217–225.
- MC CONNAUGHEY, T. (1989). C-13 and O-18 isotopic disequilibrium in biological carbonates. 1. Patterns. *Geochimica et Cosmochimica Acta* **53**, 151–162.
- MEESTERS, E. H. & BAK, R. P. M. (1995). Age-related deterioration of a physiological-function in the branching coral *Acropora palmata*. *Marine Ecology Progress Series* **121**, 203–209.
- MEZAKI, T., KESHAVMURTHY, S. & CHEN, C. A. (2014). An old and massive colony of *Pavona decussata* is sexually active at high latitude (32°N) in Japan. *Coral Reefs* **33**, 97.
- MUMBY, P. J. (1999). Bleaching and hurricane disturbances to populations of coral recruits in Belize. *Marine Ecology Progress Series* **190**, 27–35.
- NAKAMICHI, H., OJIMI, M. C., ISOMURA, N. & HIDAKA, M. (2012). Somatic tissues of the coral *Galaxea fascicularis* possess telomerase activity. *Galaxea, Journal of Coral Reef Studies* **14**, 53–59.
- NAKAMURA, T. & VAN WOESIK, R. (2001). Water-flow rates and passive diffusion partially explain differential survival of corals during the 1998 bleaching event. *Marine Ecology Progress Series* **212**, 301–304.
- NORMILE, D. (2000). Reef migrations, bleaching effects stir the air in Bali. *Science* **290**, 1282–1283.
- NOZAWA, Y. & LIN, C. H. (2014). Effects of colony size and polyp position on polyp fecundity in the scleractinian coral genus *Acropora*. *Coral Reefs* **33**, 1057–1066.
- NOZAWA, Y. & LOYA, Y. (2005). Genetic relationship and maturity state of the allorecognition system affect contact reactions in juvenile *Seriatopora* corals. *Marine Ecology Progress Series* **286**, 115–123.
- NUSSEY, D. H., BAIRD, D., BARRETT, E., BONER, W., FAIRLIE, J., GEMMELL, N., HARTMANN, N., HORN, T., HAUSSMANN, M., OLSSON, M., TURBILL, C., VERHULST, S., ZAHN, S. & MONAGHAN, P. (2014). Measuring telomere length and telomere dynamics in evolutionary biology and ecology. *Methods in Ecology and Evolution* **5**, 299–310.
- OJIMI, M. C., LOYA, Y. & HIDAKA, M. (2012). Sperm of the solitary coral *Ctenactis echinata* exhibit longer telomeres than that of somatic tissues. *Zoological Studies* **51**, 1475–1480.
- VAN OPPEN, M. J. H., SOUTER, P., HOWELLS, E. J., HEYWARD, A. & BERKELMANS, R. (2011). Novel genetic diversity through somatic mutations: fuel for adaptation of reef corals? *Diversity* **3**, 405–423.
- PERMATA, W. D. & HIDAKA, M. (2005). Ontogenetic changes in the capacity of the coral *Pocillopora damicornis* to originate branches. *Zoological Science* **22**, 1197–1203.
- PHILIPP, E. & FABRICIUS, K. (2003). Photophysiological stress in scleractinian corals in response to short-term sedimentation. *Journal of Experimental Marine Biology and Ecology* **287**, 57–78.
- PHONGSUWAN, N. & CHANSANG, H. (2012). Repeated coral bleaching in the Andaman Sea, Thailand during the last two decades. *Phuket Marine Biological Centre Research Bulletin* **71**, 19–41.
- PINZON, J. H., REYES-BONILLA, H., BAUMS, I. B. & LAJEUNESSE, T. C. (2012). Contrasting clonal structure among *Pocillopora* (Scleractinia) communities at two environmentally distinct sites in the Gulf of California. *Coral Reefs* **31**, 765–777.
- PISAPIA, C., ANDERSON, K. & PRATCHETT, M. S. (2014). Intraspecific variation in physiological condition of reef-building corals associated with differential levels of chronic disturbance. *PLoS One* **9**, e91529.
- POTTS, D. C., DONE, T. J., ISDALE, P. J. & FISK, D. A. (1985). Dominance of a coral community by the genus *Porites* (Scleractinia). *Marine Ecology Progress Series* **23**, 79–84.
- PULLI-STEPHAN, E., SENECA, F. O., MILLER, D. J., VAN OPPEN, M. J. H. & WILLIS, B. L. (2012). Expression of putative immune response genes during early ontogeny in the coral *Acropora millepora*. *PLoS One* **7**, e39099.
- REICH, H. G., ROBERTSON, D. L. & GOODBODY-GRINGLEY, G. (2017). Do the shuffle: changes in Symbiodinium consortia throughout juvenile coral development. *PLoS One* **12**, e0171768.
- REYES-BERMEDEZ, A., DESALVO, M. K., VOOLSTRA, C. R., SUNAGAWA, S., SZMANT, A. M., IGLESIAS-PIRIETO, R. & MEDINA, M. (2009). Gene expression microarray analysis encompassing metamorphosis and the onset of calcification in the scleractinian coral *Montastraea faveolata*. *Marine Genomics* **2**, 149–159.
- REYES-BERMEDEZ, A., VILLAR-BRIONES, A., RAMIREZ-PORTILLA, C., HIDAKA, M. & MIKHEYEV, A. S. (2016). Developmental progression in the coral *Acropora digitifera* is controlled by differential expression of distinct regulatory gene networks. *Genome Biology and Evolution* **8**, 851–870.
- RICHARDS, Z. T., SHEN, C.-C., HOBBS, J.-P. A., WU, C.-C., JIANG, X. & BEARDSLEY, F. (2015). New precise dates for the ancient and sacred coral pyramidal tombs of Leluh (Kosrae, Micronesia). *Science Advances* **1**, e1400060.
- RIDD, P. V., DA SILVA, E. T. & STIEGLITZ, T. (2013). Have coral calcification rates slowed in the last twenty years? *Marine Geology* **346**, 392–399.
- RINKEVICH, B., AVISHAI, N. & RABINOWITZ, C. (2005). UV incites diverse levels of DNA breaks in different cellular compartments of a branching coral species. *Journal of Experimental Biology* **208**, 843–848.
- RINKEVICH, B. & LOYA, Y. (1986). Senescence and dying signals in a reef building coral. *Experientia* **42**, 320–322.
- RINKEVICH, Y., MATRANGA, V. & RINKEVICH, B. (2007). Stem cells in aquatic invertebrates: common premises and emerging unique themes. In *Stem Cells in Marine Organisms* (eds B. Rinkevich and V. Matranga), pp. 61–103. Springer-Verlag, London.
- ROSEN, B. R. (1986). Modular growth and form of corals – a matter of metamers. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences* **313**, 115–142.
- SAENGER, C., COHEN, A. L., OPPO, D. W., HALLEY, R. B. & CARILLI, J. E. (2009). Surface-temperature trends and variability in the low-latitude North Atlantic since 1552. *Nature Geoscience* **2**, 492–495 (Letter).
- SAKAI, K. (1998). Delayed maturation in the colonial coral *Goniastrea aspera* (Scleractinia): whole-colony mortality, colony growth and polyp egg production. *Researches on Population Ecology* **40**, 287–292.
- SARETZKI, G., ARMSTRONG, L., LEAKE, A., LAKO, M. & VON ZGLINICKI, T. (2004). Stress defense in murine embryonic stem cells is superior to that of various differentiated murine cells. *Stem Cells* **22**, 962–971.
- SARETZKI, G., WALTER, T., ATKINSON, S., PASSOS, J. F., BARETH, B., KEITH, W. N., STEWART, R., HOARE, S., STOJKOVIC, M., ARMSTRONG, L., VON ZGLINICKI, T. & LAKO, M. (2008). Downregulation of multiple stress defense mechanisms during differentiation of human embryonic stem cells. *Stem Cells* **26**, 455–464.
- SCHAIBLE, R., SCHEUERLEIN, A., DANKO, M. J., GAMPE, J., MARTINEZ, D. E. & VAUPEL, J. W. (2015). Constant mortality and fertility over age in *Hydra*. *Proceedings of the National Academy of Sciences of the United States of America* **112**, 15701–15706.
- SCHUHMACHER, H., LOCH, W. & LOCH, K. (2002). Post-bleaching growth reveals *Diploastrea helianthus* to be a coral methusalem. *Coral Reefs* **21**, 344–345.
- SCHWEINSBERG, M., TOLLRIAN, R. & LAMPERT, K. P. (2016). Genetic variation in the massive coral *Porites lobata*. *Marine Biology* **163**, 242.
- SCHWEINSBERG, M., WEISS, L. C., STRIEWSKI, S., TOLLRIAN, R. & LAMPERT, K. P. (2015). More than one genotype: how common is intracolony genetic variability in scleractinian corals? *Molecular Ecology* **24**, 2673–2685.
- SCHWENTNER, M. & BOSCH, T. C. G. (2015). Revisiting the age, evolutionary history and species level diversity of the genus *Hydra* (Cnidaria: Hydrozoa). *Molecular Phylogenetics and Evolution* **91**, 41–55.
- SCOFFIN, T. P., BROWN, B. E., DUNNE, R. P. & LETISSIER, M. D. A. (1997). The controls on growth form of intertidal massive corals, Phuket, south Thailand. *PALAIOS* **12**, 237–248.
- SHARP, K. H., DISTEL, D. & PAUL, V. J. (2012). Diversity and dynamics of bacterial communities in early life stages of the Caribbean coral *Porites astreoides*. *ISME Journal* **6**, 790–801.
- SHARP, K. H., RITCHIE, K. B., SCHUPP, P. J., RITSON-WILLIAMS, R. & PAUL, V. J. (2010). Bacterial acquisition in juveniles of several broadcast spawning coral species. *PLoS One* **5**, e10898.
- SHARPLESS, N. E. & DEPINHO, R. A. (2007). How stem cells age and why this makes us grow old. *Nature Reviews Molecular Cell Biology* **8**, 703–713.
- SINCLAIR, C. S., RICHMOND, R. H. & OSTRANDER, G. K. (2007). Characterization of the telomere regions of scleractinian coral, *Acropora surculosa*. *Genetica* **129**, 227–233.
- SKÖLD, H. N. & OBST, M. (2011). Potential for clonal animals in longevity and ageing studies. *Biogerontology* **12**, 387–396.
- SOONG, K., CHEN, C. A. & CHANG, J. C. (1999). A very large poritid colony at Green Island, Taiwan. *Coral Reefs* **18**, 42.
- SOONG, K. Y. & LANG, J. C. (1992). Reproductive integration in reef corals. *Biological Bulletin* **183**, 418–431.
- SPEAKMAN, J. R., BLOUNT, J. D., BRONIKOWSKI, A. M., BUFFENSTEIN, R., ISAKSSON, C., KIRKWOOD, T. B. L., MONAGHAN, P., OZANNE, S. E., BEAULIEU, M., BRIGA, M., CARR, S. K., CHRISTENSEN, L. L., COCHEME, H. M., CRAM, D. L., DANTZER, B., et al. (2015). Oxidative stress and life histories: unresolved issues and current needs. *Ecology and Evolution* **5**, S745–S757.
- SWEET, M. J. & BROWN, B. E. (2016). Coral responses to anthropogenic stress in the twenty-first century: an ecophysiological perspective. In *Oceanography and Marine Biology: An Annual Review*, Volume 54. *Oceanography and Marine Biology* (eds R. N. Hughes, D. J. Hughes, I. P. Smith and A. C. Dale), pp. 271–314. CRC Press-Taylor & Francis Group, Boca Raton FL, USA.
- SWEET, M. J., BROWN, B. E., DUNNE, R. P., SINGLETON, I. & BULLING, M. (2017). Evidence for rapid, tidal-related shifts in the microbiome of the coral *Coelastrea aspera*. *Coral Reefs* **36**, 815–828.

- SZMANT, A. M. (1986). Reproductive ecology of Caribbean reef corals. *Coral Reefs* **5**, 43–53.
- TSUTA, H. & HIDAKA, M. (2013). Telomere length of the colonial coral *Galaxea fascicularis* at different developmental stages. *Coral Reefs* **32**, 495–502.
- TSUTA, H., SHINZATO, C., SATOH, N. & HIDAKA, M. (2014). Telomere shortening in the colonial coral *Acropora digitifera* during development. *Zoological Science* **31**, 129–134.
- VASQUEZ-BEDOYA, L. F., COHEN, A. L., OPPO, D. W. & BLANCHON, P. (2012). Corals record persistent multidecadal SST variability in the Atlantic Warm Pool since 1775 AD. *Paleoceanography* **27**, PA3231.
- VAUPEL, J. W., BAUDISCH, A., DOLLING, M., ROACH, D. A. & GAMPE, J. (2004). The case for negative senescence. *Theoretical Population Biology* **65**, 339–351.
- VOOLSTRA, C. R., SCHWARZ, J. A., SCHNETZER, J., SUNAGAWA, S., DESALVO, M. K., SZMANT, A. M., COFFROTH, M. A. & MEDINA, M. (2009). The host transcriptome remains unaltered during the establishment of coral-algal symbioses. *Molecular Ecology* **18**, 1823–1833.
- WALLACE, C. C. (1999). *Staghorn Corals of the World – A Revision of the Genus Acropora*. CSIRO Publishing, Collingwood.
- WEIS, V. M. (2008). Cellular mechanisms of Cnidarian bleaching: stress causes the collapse of symbiosis. *Journal of Experimental Biology* **211**, 3059–3066.
- WHITE, R. R., MILHOLLAND, B., DE BRUIN, A., CURRAN, S., LABERGE, R. M., VAN STEEG, H., CAMPISI, J., MASLOV, A. Y. & VIJG, J. (2015). Controlled induction of DNA double-strand breaks in the mouse liver induces features of tissue ageing. *Nature Communications* **6**, 6790.
- WILLIAMS, A. D., BROWN, B. E., PUTCHIM, L. & SWEET, M. J. (2015). Age-related shifts in bacterial diversity in a reef coral. *PLoS One* **10**, e0144902.
- VAN WOESIK, R., IRIKAWA, A., ANZAI, R. & NAKAMURA, T. (2012). Effects of coral colony morphologies on mass transfer and susceptibility to thermal stress. *Coral Reefs* **31**, 633–639.
- YEOH, S. R. & DAI, C. F. (2010). The production of sexual and asexual larvae within single broods of the scleractinian coral, *Pocillopora damicornis*. *Marine Biology* **157**, 351–359.
- ZAPATA, F., GOETZ, F. E., SMITH, S. A., HOWISON, M., SIEBERT, S., CHURCH, S. H., SANDERS, S. M., AMES, C. L., MCFADDEN, C. S., FRANCE, S. C., DALY, M., COLLINS, A. G., HADDOCK, S. H. D., DUNN, C. W. & CARTWRIGHT, P. (2015). Phylogenomic analyses support traditional relationships within Cnidaria. *PLoS One* **10**, e0139068.
- ZIELKE, S. & BODNAR, A. (2010). Telomeres and telomerase activity in scleractinian corals and *Symbiodinium* spp. *Biological Bulletin* **218**, 113–121.

(Received 12 July 2017; revised 17 November 2017; accepted 23 November 2017; published online 28 December 2017)